

VITAMIN D DEFICIENCY AND CORONARY HEART DISEASE

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Coronary heart disease (CHD) is the result of the accumulation of atheromatous plaques within the walls of the arteries that supply the myocardium with oxygen and nutrients. The WHO estimated that in 2002, 12.6% of deaths worldwide were from CHD.

The active form of vitamin D, calcitriol, exerts genomic and nongenomic effects through a cytosolic vitamin D receptor (VDR) and a membrane bound receptor. VDRs have been found in almost all human tissues and cells, among them cardiomyocytes, endothelial cells, and vascular smooth muscle cells. Now there is increasing evidence that calcitriol exerts important physiological processes in the vasculature. These mechanisms include the inhibition of vascular smooth muscle proliferation, the suppression of vascular calcification, the down regulation of pro-inflammatory cytokines, the up regulation of anti-inflammatory cytokines, the action of vitamin D as a negative endocrine regulator of the renin–angiotensin system, and the inhibition of vascular calcification. Vascular calcification is an important risk factor for CVD mortality in the general population and is a frequent finding in patients with CVD. Data from two human populations at high and moderate risk for ischemic heart disease indicate an inverse association of serum calcitriol levels with vascular calcification.

During the last decade, it became clear that deficiency of serum concentrations of vitamin D metabolites are prevalent not only in specific patient groups but also in the general population in western countries and throughout the world. The much more important cause of this phenomenon is an inadequate skin exposure to solar ultraviolet B radiation, as ultraviolet B induced skin synthesis is the major source of vitamin D for humans. Ecological studies have reported higher rates of CHD with increasing distance from the equator, a phenomenon that can be attributed to the higher prevalence of vitamin D deficiency in regions with less exposure to sunlight.

Vitamin D deficiency could be an additional risk factor in the etiology and progression of CHD.

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